the minor forms might be employed with direct advantage. He had one case in which a condition of hysterical hemianæsthesia had resulted from a fright, the patient believing he had received the electric current. This case had been successfully treated by hypnotism and hydrotherapy, and speaker attached a good deal of importance to the water part of the treatment.

DR. W. R. BIRDSALL thought that in the case under consideration it was a point to bear in mind how very slight blows upon the shoulder were frequently the origin of atrophy of the deltoid.

AMERICAN NEUROLOGICAL ASSOCIATION.

Dr. WHARTON SINKLER, Pres.; Dr. G. M. HAMMOND, Sec.

Seventeenth Annual Meeting, held at Washington, D. C., September 22, 23, and 24, 1891.

(*Continued.*)

DOUBLE ATHETOSIS.

DR. WILLIAM OSLER, of Baltimore, read a paper on this subject, based upon fifty-three cases in literature and numerous personal observations. He mentioned incidentally that this condition is compatible with a high degree of intelligence, but is usually accompanied by mental deterioration.

LESIONS OF THE SUPERIOR TEMPORAL CONVOLUTIONS ACCURATELY LOCATING THE AUDITORY CENTRE.

By CHARLES K. MILLS, M.D.

Although the localization of the auditory centre or sphere in the first or in the two upper temporal convolutions is generally admitted, this view has not received universal acceptance ; therefore, the great value of the history and autopsy here recorded will be acknowledged.

The patient, a woman, aged 46 years, was admitted to the Philadelphia Hospital in August, 1891. She had a history of scarlet fever in childhood, of rheumatism many years ago, and also of old venereal disease. She had had five abortions or miscarriages, and had one living child, 23 years of age, who was deaf and weakminded. For many years she had had valvular disease of the heart. She was right-handed. Some of the facts of her history were obtained from relatives who called to see her at the hospital, and others were procured after her death from a brother and sister-in-law, who were sought out and carefully interviewed.

Fifteen years before her death she had an apoplectic attack which left her word-deaf but not paralyzed. Prior to this first attack of apoplexy her hearing had been good, but after it she could not, by hearing, understand anything that was said to her. She could, however, hear music and sounds of various kinds; for instance, when an organ or band had performed upon the street she at times had called attention to the fact; and she had also come down from the second or even the third story to open the front door in answer to a knock. She could hear such sounds as a bell ringing and a clock ticking. These facts were elicited from her relatives through various statements made by them, chiefly spontaneously.

When anyone wished to communicate with her it was done by means of writing or signs, as she had fully preserved her vision and was evidently not word-blind either for writing or printing. She often read the newspapers, and could do so with intelligence up to a few weeks before her death. Her sister-in-law said that several times she had heard her try to read the newspaper aloud, and in so doing she had seemed to understand fully what she read, "but made a tangle of her words." From the time of the first attack she had never been able to speak well, her words becoming "jumbled" or "tangled." From the description given of her manner of speech the defect was evidently a serious form of paraphasia and paralexia.

Her relatives spoke positively of her deafness as having been due to the "stroke;" but the apoplectic attack, although it had at once caused this word-deafness and paraphasia, had not in any way, as far as could be ascertained, affected either motion or sensation. She could write, but "sometimes mixed up her words in writing."

Nine years before her death she had another and more severe apoplexy, after which her deafness increased for sounds as well as words until it was almost total. This seizure left her also with partial left hemiplegia, chiefly affecting the arm, and in this extremity, from the description, the paralysis was more marked below the elbow. Upon examination after admission to the hospital, she was found to have some contracture at the metacarpo-phalangeal articulation.

Six weeks before her admission she began to have pain in the left side and abdomen, which gradually increased in severity. The day before her admission she had a chill and pain in the left shoulder and arm. For five weeks she had involuntary evacuations of urine, and for about two weeks she had been unable to retain her fæces. For these six weeks she had been much more helpless than previous to this time, often letting things drop from her hands. She seemed to suffer considerably from headache, and also slept badly.

She was first examined by me on August 24, 1891. Her condition then was one of almost complete helplessness. It was impossible to make her understand what was said to her, and so far as could be determined by repeated tests she was totally deaf; but notwithstanding her weakness, helplessness, and deafness, her face had a somewhat intelligent expression. She looked about her as if she knew what was going on. She was very emaciated; her heart's action was excited, and examination showed the presence of marked murmurs, both mitral and aortic. She became feebler day by day, and died August 28.

Autopsy.—No disease of the bone or of the dura mater was found. Even before pia-arachnoid of the left hemisphere was removed, it was noticeable that the first temporal convolution was much smaller and thinner than usual, and that at the posterior extremity of it and of the second temporal was a depression covering a space about seven-eighths of an inch in diameter.

The left first temporal convolution is remarkably small, narrow and smooth, except at its anterior extremity. Its posterior two-thirds or three-fourths have shrunken to a thin strip. At a point about the middle of the gyre, the convolution has so disappeared as to leave only a notch and shred of tissue. Just anterior to this point a small annectant gyre runs to the second temporal. The attenuated appearance of this superior temporal convolution is such as to attract attention of the most inexperienced observer of such appearances.

At a position marked, corresponding to the posterior fourth of the second temporal convolution and the parallel fissure, the brain presented a marked depression or cavity, at the bottom of which, when the specimen was in the fresh state, was a small mass of yellow, shrivelled, puckered tissue. This was evidently the remains of an old, embolic softening.

The subarachnoid cavity or cyst, which was present before the inner membranes were removed, was formed by this old necrosed area and the widened parallel fissure, this widening having chiefly resulted from the atrophying of the first temporal convolution.

In connection with the question of the part played by the second temporal convolution in cerebral audition, it is, of course, important to carefully describe the condition of this convolution. From the position of the annectant convolution seen in the figure backward it is decidedly atrophied, and in its posterior fourth, or perhaps third, it has practically disappeared, and has been replaced by the cavity or cyst just described.

The third, fourth and fifth temporal convolutions were undoubtedly not involved in either the softening or atrophy, as the brain was carefully studied by me in both directions, from the second temporal convolution to the hippocampal and the reverse.

Around the ascending branch of the Sylvian fissure and at the bases of the two central convolutions also, much atrophy had evidently also taken place. This ascending branch of the Sylvian, instead of being a mere indentation or narrow fissure as is usually the case, is wide and gaping. The hinder portion of the third frontal, and particularly the strip of convolution between the ascending Sylvian and precentral fissure, is markedly wasted.

The retro-insular convolutions are two in number, and the posterior of the two is very small. The anterior retro-insular presents the appearance of being a continuation of the anterior half of the first temporal, and the posterior retro-insular is continuous with the posterior much-shrunken half of the first temporal.

In the right hemisphere is an old and very extensive hemorrhagic cyst, which, as shown by the specimen and the photograph, has completely destroyed the first, and almost completely the second, temporal gyre, the island of Reil, retro-insular gyres, the lower extremities of the central gyres, and a large extent—but exactly how much could not be determined—of the ganglia and capsules. Examination from within showed that the caudate body and the thalamus had largely preserved their integrity, and the chief interior destruction was probably of the lenticular ganglion and the external capsule. In the posterior portion of the cystic area, some gyral substance is seen, but examination shows this to be the sunken-in lower extremity of the inferior parietal convolution. As on the other side, the supra-Sylvian bordering convolutions are much atrophied. The auditory nerves were atrophied, The acoustic striæ, usually so easily seen, and often so prominent, could not be made out with the naked eye.

Slight adhesions of the left pleura were present on both sides. The lungs were œdematous and exuded a turbulent fluid on pressure. The pericardial sac contained an ounce and a half of fluid. The endocardium was thickened. The mitral valve was also much thickened, the orifice being so small that it would not admit the little finger. The aortic valves were in a similar but worse condition, and a clot was found in the left auricle resembling in appearance those seen in aneurisms. The spleen was small. The kidneys were small and presented on their surface large hemorrhagic spots. On section the surface was found to be flabby, and the cortex and pyramids much distorted. The spots on the surface had irregular shapes, having the appearance of old and recent hemorrhagic infarcts. The liver was small and moderately firm. The ovaries were dense and sclerotic.

A study of this case justifies the following conclusions :

(1) The centre for word-hearing is situated in the hinder thirds of the first and second temporal convolutions ; its exact position is in a line with, or just in front of, the posterior extremity of the horizontal branch of the fissure of Sylvius. Possibly it is restricted to the second temporal convolution.

(2) The third, fourth and fifth temporal convolutions take no part in cerebral audition.

(3) A lesion confined to the posterior thirds of the first and second temporal convolutions of the left hemisphere will produce complete, or almost complete, word-deafness, the corresponding regions of the other hemisphere remaining intact.

(4) The field or sphere for all auditory memories covers a much larger cortical area than that for word-hearing, including at least the posterior two-thirds of the first and second temporal convolutions.

(5) The auditory field and special auditory centres have their highest development in the left hemisphere, but destruction of the auditory areas of the two upper temporal convolutions of both hemispheres is necessary to complete brain deafness.

(6) A lesion limited to the centre for word-hearing and causing word-deafness will cause also paraphasia in attempts at speaking, and paralexia in attempts at reading.

(7) An isolated lesion of the centre for word-hearing, producing absolute, or nearly absolute, word-deafness, does not necessarily cause inability to recall words by other means, as, for instance, through their visual signs ; in such cases probably the meaning of the word is understood, although the name cannot be properly verified in consciousness.

(8) A cerebral lesion or lesions causing word-deafness will, in time, lead to secondary atrophy of the speech and oro-lingual centres on the motor or emissive side of the brain, and also to atrophy of the association tracts between the sensory and motor-hearing-speech centres.

(9) The retro-insular convolutions are anatomically and functionally closely related with subdivisions of the first temporal convolution, the most posterior of these retro-insular convolutions being continuous with the posterior half or two-thirds of the first temporal convolution.

Subcortical Hemorrhagic Cyst Beneath the Arm and Leg Areas.—DR. MILLS reported another case with the above title. (See page 803.)

Autopsy on a Case of Athetoid Spasm Myotonia, and Diffuse Bilateral Disturbances of Sensation; Chronic Convexity Meningitis of both Hemispheres with Cortical and Subcortical Softening; Lesions being most marked in the Posterior Parietal Region.—The same writer reported an additional case with the above title. (See page 794.)

TUMOR OF THE MID-BRAIN AND LEFT OPTIC THALAMUS.

DR. JAMES H. LLOYD, of Philadelphia, reported the case and exhibited the specimen from a patient twenty-eight years of age, who had been admitted to the Philadelphia Hospital. There was a crossed paralysis, the left third nerve being involved, with right brachial monoplegia and right crural monoparesis. The face and tongue were not paralyzed on either side. No paralysis of any other cranial nerve. Death took place on the twentieth day after admission. The autopsy revealed a tumor of the left optic thalamus and the mid-brain, involving the left cerebral peduncle. On microscopical investigation this was found to be a glioma. A diagnosis of tumor of the left cerebral peduncle had been made at the first examination.

DR. F. X. DERCUM said that the sensory features in Dr. Mills' cases would prove an interesting study.

DR. J. J. PUTNAM referred to the autopsy, in which no lesion could be found, in the case of a child with double athetosis. In a case of hemichorea there was a distinct spot of softening in the tegmentum on one side. In another patient with locomotor ataxia and no impairment of intelligence, there was a linear subcortical hemorrhage, in the subcortical area below the paracentral lobule. Secondary degeneration had occurred in the cord. He also spoke of a case of sensory aphasia that was operated upon, with the result of finding a cyst occupying the left temporal lobe.

DR. L. C. GRAY mentioned the case of a man with hemiplegia and impairment of the muscular sense, in whom nothing could be discovered on operation. Death took place on the following day. The autopsy revealed a round-cell sarcoma with cystic degeneration in its centre, at the junction of the arm and leg centres. This was situated in the subcortex. There was also a sarcoma of the thigh.

DR. MILLS asked if the paralysis was spastic.

DR. GRAY replied that it was not, but he thought that spastic symptoms generally occurred where the lesion was near the basal ganglia.

DR. C. L. DANA cited the case of a man aged forty-six, who had syphilis ten years before. There were headache, left hemi-convulsions and coma. He recovered with left hemiplegia and left hemianæsthesia. The autopsy showed a gumma of the dura over the right central convolutions. In another case of a man aged fifty-five, with progressive right hemiplegia, and some loss of sensibility on the same side, there was a tumor about the size of a hen's egg in the middle of the left precentral convolution. He also narrated the histories he had collected of several cases of similar character, showing localization of sensory centres. He held the view that the cutaneous sensations must have their representation in the brain cortex, and had a larger and more diffuse representation than the motor. When one side was affected, compensation would take place from the other side to a greater extent than in motion or other sensations. He claimed that there is no evidence to prove the contrary. Dr. Mills' claim that the centre for cutaneous sensation is in the parietal lobe was at variance with all clinical facts.

DR. KNAPP reported the autopsy of a case of cerebral infantile paralysis with hemiathetosis. The child had hydrocephalus and tubercular basilar meningitis. The microscopic examination of the cord was negative, but the specimen was not a good one, and the result of the examination was therefore uncertain. He believed that the auditory centres were in the temporal lobe, and he regarded Dr. Mills' case as one of the most conclusive yet reported. It is probable that each temporal lobe contains representations for both ears, and hence we cannot determine accurately the auditory centre, until we have methods of testing the hearing as complete as those we have for testing the vision. The rare cases of bilateral lesion of the temporal lobes are therefore the only ones which give us any definite information as to the auditory centres in man. His views as to sensory representation in the cortex agreed with those of Dr. Dana. Cases undoubtedly occurred where there was lesion in the Rolandic region without sensory disturbance, but cases had also occurred of lesion in that region without motor disturbance, notably a case of tumor, reported by Bramwell. He had reported a striking case of lesion in the ascending parietal, with sensory disturbance, at the last meeting of the Association, but Dr. Mills did not accept it as conclusive. In that case, as in a case of tumor of Rolandic region, there was no autopsy to determine absolutely the seat of the injury. Ferrier denied that the visual centre was in the cuneus, and Schaefer that the auditory centre was in the temporal lobe, and they both denied that the sensory centres were in the Rolandic region ; yet Dr. Mill's case had anæsthesia without lesion of Ferrier's sensory centre in the gyrus fornicatus. He agreed with Dr. Dana that tactile sensibility was more fully represented back of the fissure of Rolando than in front of it.

DR. J. W. PUTNAM spoke of a case where, shortly after the removal of nasal polypi, there was gradual loss of memory, and at the eighth day, owing to the absence of sensory or motor disturbance, the diagnosis was made of abscess of the left frontal lobe. Trephining was done, and three ounces of pus removed from the frontal lobe. At the autopsy there was found an abscess of the corresponding frontal lobe. In another case with spastic condition of all four extremities, there was a glioma at the base, pushing the medulla to one side. In a child with athetosis involving the head as well as all the extremities, he found apparent absence of the bridge of the corpus callosum, and an abscess in the apex of each temporal lobe. The case had been diagnosed by a good observer as absence or defect of the cerebellum.

DR. MILLS said he had seen a case where tactile sensibility was lost in the hand, after loss of brain-substance from the Rolandic region. This was the result of a fractured skull. He thought it would be difficult to locate the cortical areas in the negro by the same measurements in general use.

DR. J. J. PUTNAM referred to a case where the accidental removal of part of the central convolution occurred during operation, and was followed by sensory disturbance.

DR. BREMER said that he had seen a case in which an attempt was made to excise the wrist centre on the right side for focal epilepsy. Neither paralysis nor other symptoms followed the operation. He considered the question unsettled as to cortical motor and sensory areas. In the presence of spastic symptoms the question is not so much as to the location of the lesion in the subcortex, but the character of the tumor. The varying amount of blood-supply in a hemorrhagic cyst, probably gives rise to spastic symptoms.

DR MILLS, in closing the discussion, said we are not yet in a position to settle this matter of sensory localization. He thought his case was not conclusive. His view is not of Ferrier, Horsley, or Munk, but that we have a great sensory lobe, not the gyrus fornicatus or the hippocampal region, but an anatomically demarcated lobe, which may include the gyrus fornicatus, hippocampal convolution, quadrate and posterior parietal lobule. In these regions are subdivisions of this sensory lobe, corresponding to subcutaneous muscular groups. Dr. Dana admits that in the cortex sensation has more representation than motion. This suggests one argument in settlement. Dana's cases must be satisfactorily explained. He could give many cases to Dana's one case, in substantiation of his view. There must be a sensory-motor association tract as well as a sensory-motor audition tract; cerebral paræsthesia comparable with paraphasia and paralexia. The convincing cases will be the bilateral. If this be true, then Dana has nothing to substantiate his claim. He suggested that sensation can be inhibited, as illustrated by hypnotism. Possibly we may explain the sensory symptoms by inhibition by tumor in the motor region of the adjacent sensory region. He claimed that operative cases showed no objective sensory symptoms after the removal of large portions of the motor cortex. He was surprised to hear it said that men who supported sensory localization did not support the correct views as to the centre for audition. As to myotonia and athetoid movements in his case, he was confident that there was no lesion in the capsule and no softening anterior to the retrocentral fissure.

THOMSEN'S DISEASE.

Dr. Walton then read a paper describing a typical case of Thomsen's disease.

The patient was a man of twenty-eight, who had suffered from the rigidity peculiar to this disease from infancy. As a boy his movements were clumsy and slow, although he could endure fatigue as well as any when once the muscles were brought into play. His condition has been at a standstill since he was fifteen. On rising it is difficult to set the limbs in motion, and the gait is at first stiff and waddling, somewhat as if he were drawing his feet from the mud. Once under way he can walk many miles rapidly and without fatigue. All muscles are similarly affected, including those of deglutition, and the eyelids, which he finds it hard to drop after looking up. Individual movements are fairly strong. The muscles are large and firm. The position in standing resembles that of pseudo-muscular hypertrophy (photograph shown), this disease is, however, excluded by the course of the case, and by the strength of the muscles whenever brought into use. There is increased mechanical irritability, and the faradic current produces tonus. The characteristic reaction to the galvanic current could not be obtained, the responses being normal to both strong and weak currents. There was no further family history beyond a marked neurotic taint, and the fact that the father had suffered similarly, but in very light degree, in early life.

A case of Eulenburg's paramyotonia which had recently come under the writer's observation was reported briefly to show the similarity and difference between the two conditions. The case was that of a gentleman sixty-seven years of age, who was subject to tonic spasm, affecting principally the abdomen and lower extremities, coming on after exercise, lasting from a quarter to half an hour. On continuance of exercise the spasm reappears, after which the patient can continue without trouble. The difficulty is congenital, and was present in a younger brother and a daughter.

Both diseases are due to congenital defect in the muscular apparatus, perhaps also in the central nervous system, and are probably little affected, if at all, by treatment.

Dr. Angell, of Rochester, read the history of two similar cases. See page 807.

Dr. Hughes maintained that Thomsen's disease is an affection of the muscular system, and doubted the entity of such a disease. Thinks it a peculiar form of symptomatic condition. It differs from every kind of spasm with which we are familiar.

THE ELECTRO-PHYSIOLOGY OF REFLEXES, WITH THE DESCRIPTION OF A HITHERTO UNKNOWN LOCALIZED PHYSIOLOGICAL REFLEX PHENOMENON.

DR. GEORGE W. JACOBY read a paper on this subject (accompanied by a demonstration), in which he said that our knowledge of reflexes produced by electrical excitation is still in its infancy, and an electro-physiology of reflexes does not yet exist. The reasons assigned for this lack of knowledge are the necessity of employing very strong currents, and the difficulty of deciding whether contractions thus produced are reflex or not; these reasons he considers entirely invalid. After numerous experiments he discovered a reflex, produced by the application of the negative pole of a galvanic battery to the radial side of the forearm, which consisted in a contraction of muscles of the chin.

Examination of two hundred persons showed that the reflex was present in over seventy per cent. of normal individuals. The experiments were conducted by placing a large Erb electrode over the back of the hand and then making closure of the current with a small electrode attached to an interrupting handle. All such experiments upon the human body have heretofore been entirely dependent upon the statements and feelings of the patient. These unreliable factors can now be eliminated by utilizing the reflex described, as hereby we have a visible proof of the sensory reaction. Jacoby does not doubt that, attention having now been called to this reflex, other reflexes of a similar nature may be found in various parts of the body, and that we will by their examination obtain knowledge not only in regard to the pathological changes in sensory nerves, but also in regard to changes in that part of the cord upon whose integrity the excitation of such reflexes depends.

SUPPLEMENTAL TREATMENT OF THE PARALYSIS OF ACUTE ANTERIOR POLIO-MYELITIS.

DR. V. P. GIBNEY read a paper on this subject, and said that a palsied muscle, or a group of palsied muscles, is often very much handicapped by contractured tendons or muscles on the opposite side of the limb. Because of these contractures local treatment is often inefficient. However efficient the galvanic or the faradic current, or even massage, may be, if the muscle is overstretched the efficiency is certainly minimized. Another point was this, that a palsied muscle, or a group of palsied muscles, responds better to local treatment after a period of rest, or at least after being retained for a long time in normal position. In this connection he reported the histories in six cases. He advocated the correction of deformity, the removal of tension from a weakened muscle, the replacement of the joint surfaces to normal apposition, and the assistance generally given to a limb, the muscles of which are weak and palsied.

INTRA-CRANIAL SYPHILIS.

DR. LANDON CARTER GRAY read a paper upon the diagnosis of a certain form of intra-cranial syphilis, giving the histories of a number of cases in support of his views. These symptoms were a cephalalgia that is quasi-periodical, recurring generally at night, occasionally in the afternoon or in the morning, with marked insomnia ; this cephalalgia and insomnia ceasing suddenly upon the supervention of any paralytic symptoms. Dr. Gray also called attention to the fact that a hemiplegia occurring in an individual under middle age should render us very suspicious of a syphilitic causation. This peculiar headache and insomnia belonged to the early stage of intra-cranial syphilis, although it might occur in the primary, secondary, or tertiary state of the general syphilitic infection.

DR. MILLS said that the occurrence of paralysis after the subsidence of headache and insomnia was an old observation but not for that reason an unimportant feature in the diagnosis or treatment of syphilis. These symptoms occurred in consequence of degenerative changes.

DR. J. J. PUTNAM agreed with Dr. Mills, and mentioned three cases illustrating the supervention of paralysis on the cessation of headache and insomnia. He believed that anæmia or arterial congestion gave rise to the former condition, thus rendering the patient less sensitive to pain.

DR. HUGHES said that the persistency of the headache was of more significance than its periodicity.

SEVEN RECENT CASES OF BRAIN SURGERY.

DR. WILLIAM A. HAMMOND presented the history of seven cases in which he had operated for the relief of epilepsy or paralysis, and said that the cases were of somewhat different character, for not only was the seat of a real or supposed injury a factor in determining where to operate, but the symptoms, their nature and location, were even of more importance in deciding upon the situation of the disease, the consequent point on the skull with which it was in relation and the extent to which the operation should be carried.

TUBERCULAR INFECTION OF THE CENTRAL NERVOUS SYSTEM.

DR. B. SACHS read a paper with this title. In the two cases serving as a basis for the paper, the patients had been received into the "Montefiore Home" for a non-tubercular disease of the central nervous system. In the one case, a large hemorrhagic cyst in the cortex was the residue of the process, giving rise to an infantile spastic hemiplegia in the vicinity of this cyst. Soon after admission to the "Home," where there were many patients with phthisis, a solitary tubercle of enormous size was developed, which led to a rapidly fatal termination.

In the second case, a tubercular infection hastened the course of an old chronic myelitis. The history of this case is about as follows :

J. R——, a Russian, aged sixty ; family history entirely negative ; well until two years and a half ago ; then, after an exposure to cold, noticed a weakness of both legs ; greater in the left than the right ; this paresis gradually increased, associated with slight vesical symptoms. No change in symptoms for some months ; then quite suddenly, about five months after his exposure to the phthisical contagion, he became absolutely paraplegic, and the reflexes of the lower extremities increased ; bladder symptoms and bedsores are developed, the paralysis extends upward, first to the right arm and then to the left ; anæsthesia as far as navel from below upward, and gradually extending to the lower margin of the second rib ; patient has high fever, becomes rapidly emaciated, and dies about three weeks after onset of the absolute paraplegia.

Post-mortem examination : Entire absence of brain lesions, no tubercular meningitis anywhere ; but the most widespread destruction of the cord from the second to the twelfth dorsal segments, with ascending and descending degenerations. In the mass of destroyed tissues the tubercle baccilli were found in large numbers.

Dr. Sachs insists on the fact that the preceding disease of the cord furnished an excellent soil for the growth of the bacilli, and instead of growing in the lung tissues, or even in the meninges, they have grown upon the diseased substance of the cord itself. It is proper, therefore, to speak of a tubercular invasion.

Dr. Hughes referred to the unusually rapid termination of these cases with tubercular infection.

LEAD-POISONING, WITH SPECIAL REFERENCE TO THE SPINAL CORD AND TO PERIPHERAL NERVE-LESIONS.

Dr. E. D. Fisher read a paper on the above subject and gave the following report: A. E., æt. thirty-four, painter; patient first seen June, 1890; gave history of an attack of lead colic with some weakness of the hands thirteen years previous, followed two years later by a second attack; from that time on several mild attacks; continued, however, at his work until he was unable to hold his brush; March, 1890, had an epileptic seizure, losing consciousness.

Present condition: Patient anæmic; complains of weakness of forearms and hands; no sensory disturbance; examination showed marked atrophy of the muscles of the forearms and of the hands, the latter presenting the appearance of progressive muscular atrophy; slight loss of sensation to temperature, pain and touch.

Electrical examination: Loss of faradic excitability in the interossei muscles and the opponens pollicis; extensors of the forearm give slight response; the supinators were unaffected; no reaction of degeneration; heart hypertrophied; high tension of radial artery; urine: sp. gr. 1008; albumen.

Ophthalmoscopic examination: R. E. $\frac{20}{30}$; L. E. $\frac{20}{100}$; R. E. exudative neuro-retinitis; L. E. atropy of optic nerve and retina with occlusion of some of the vessels; suspected degenerative nephritis.

Patient died March, 1891, from advanced Bright's disease.

Autopsy: Heart hypertrophied; lungs normal; kidneys seat of interstial nephritis; arteries of the brain atheromatous.

Microscopical examination of the cord and muscular spinal nerve showed the following results :

The peripheral nerve markedly degenerated. There was atrophy of the anterior horn on one side and sclerosis of the antero-lateral column of that side. In certain levels there was sclerosis of the column of Goll, and in Lissauers zone there was increased connective tissue, with thickening of the meninges and the walls of the vessels.

This case, with others, that have been reported would seem to prove that the various toxic agents affect the central nervous system as well as the peripheral. Indeed, we may have them individually involved, or in combination, or again simultaneously. The affection is usually a diffuse one, not limiting itself to the special sensory or motor tracts as in the so-called systemic diseases, but involving in different areas all the columns of the cord.

DR. DANA had seen the specimens, but was unwilling to say the lesions were due to lead. Lead seems to possess a selective action for the peripheral nerves. He thought the changes more marked in the nerves than in the cord.

DR. RIGGS, of St. Paul, believed with Dr. Dana that lead usually affected the peripheral nerves, but in this case he thought the lesion was more likely due to arterial sclerosis.

DR. LLOYD then showed microscopic sections from a case of lead-poisoning that had developed locomotor ataxy.

DR. DANA presented, in behalf of Dr. Joseph Collins, two new methods of Weigert for staining specimens.